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Wi-Fi is an important threat to human health☆

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ABSTRACT

Repeated Wi-Fi studies show that Wi-Fi causes oxidative stress, sperm/testicular damage, neuropsychiatric effects including EEG changes, apoptosis, cellular DNA damage, endocrine changes, and calcium overload. Each of these effects are also caused by exposures to other microwave frequency EMFs, with each such effect being documented in from 10 to 16 reviews. Therefore, each of these seven EMF effects are established effects of Wi-Fi and of other microwave frequency EMFs. Each of these seven is also produced by downstream effects of the main action of such EMFs, voltage-gated calcium channel (VGCC) activation. While VGCC activation via EMF interaction with the VGCC voltage sensor seems to be the predominant mechanism of action of EMFs, other mechanisms appear to have minor roles. Minor roles include activation of other voltage-gated ion channels, calcium cyclotron resonance and the geomagnetic magnetoreception mechanism. Five properties of non-thermal EMF effects are discussed. These are that pulsed EMFs are, in most cases, more active than are non-pulsed EMFs; artificial EMFs are polarized and such polarized EMFs are much more active than non-polarized EMFs; dose-response curves are non-linear and non-monotone; EMF effects are often cumulative; and EMFs may impact young people more than adults. These general findings and data presented earlier on Wi-Fi effects were used to assess the Foster and Moulder (F&M) review of Wi-Fi. The F&M study claimed that there were seven important studies of Wi-Fi that each showed no effect. However, none of these were Wi-Fi studies, with each differing from genuine Wi-Fi in three distinct ways. F&M could, at most conclude that there was no statistically significant evidence of an effect. The tiny numbers studied in each of these seven F&M-linked studies show that each of them lack power to make any substantive conclusions. In conclusion, there are seven repeatedly found Wi-Fi effects which have also been shown to be caused by other similar EMF exposures. Each of the seven should be considered, therefore, as established effects of Wi-Fi.

1. Introduction

Wi-Fi (also known as WiFi or WLAN) is a wireless network involving at least one Wi-Fi antenna connected to the internet and a series of computers, laptops and/or other wireless devices communicating wirelessly with the Wi-Fi antenna. In this way, each such wireless communication device can communicate wirelessly with the internet. All the studies reviewed here were of Wi-Fi using the 2.4 GHz band, although there is also a 5 GHz band reserved for possible Wi-Fi use.

Telecommunications industry-linked individuals and groups have claimed that there are no and cannot possibly be any health impacts of Wi-Fi (Foster and Moulder, 2013; Berezow and Bloom, 2017). However with Wi-Fi exposures becoming more and more common and with many of our exposures being without our consent, there is much concern about possible Wi-Fi health effects. This paper is not focused on anecdotal reports but rather on 23 controlled, scientific studies of such health-related effects in animals, cells including human cells in culture and in human beings (Table 1).

Each of the effects reported above in from 2 to 11 studies, have an extensive literature for their occurrence in response to various other non-thermal microwave frequency EMFs, discussed in detail below. These include (see Table 1) findings that Wi-Fi exposures produce impacts on the testis leading to lowered male fertility; oxidative stress; apoptosis (a process that has an important causal role in neurodegenerative disease); cellular DNA damage (a process causing cancer and germ line mutations); neuropsychiatric changes including EEG changes; hormonal changes.

The discussion here focuses on those Wi-Fi effects which have been found by multiple Wi-Fi studies and have been previously confirmed by non-thermal exposures to other microwave frequency EMFs. The 1971/72 U.S. Office of Naval Medical Research study (Glaser, 1971) reported the following changes related to testis or sperm: 1. Decreased testosterone leading to lowered testis size. 2. Histological changes in testicular epithelial structure. 3. Gross testicular histological changes. 4.

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Table 1
Summary of health impacts of Wi-Fi EMF exposures.

Citation(s)	Health Effects
Atasoy et al. (2013); Özorak et al. (2013); Aynali et al. (2013); Çiftçi et al. (2015); Tök et al. (2014); Çiğ and Nazıroğlu (2015); Ghazizadeh and Nazıroğlu (2014); Yüksel et al. (2016); Othman et al. (2017a, 2017b); Topsakal et al. (2017)	Oxidative stress, in some studies effects lowered by antioxidants
Atasoy et al. (2013); Shokri et al. (2015); Dasdag et al. (2015); Avendaño et al. (2012); Yildiring et al. (2015); Özorak et al. (2013); Oni et al. (2011); Akdag et al. (2016)	Sperm/testicular damage, male infertility
Papageorgiou et al. (2011); Maganioti et al. (2010); Othman et al. (2017a, 2017b); Hassanshahi et al. (2017)	Neuropsychiatric changes including EEG; prenatal Wi-Fi leads to post-natal neural development, increased cholinesterase; decreased special learning; Wi-Fi led to greatly lowered ability to distinguish familiar from novel objects, changes in GABA and cholinergic transmission
Shokri et al. (2015); Dasdag et al. (2015); Çiğ and Nazıroğlu (2015); Topsakal et al. (2017)	Apoptosis (programmed cell death), elevated apoptotic markers
Avendaño et al. (2012); Atasoy et al. (2013); Akdag et al. (2016)	Cellular DNA damage
Saili et al. (2015); Yüksel et al. (2016); Topsakal et al. (2017)	Endocrine changes incl.: Catecholamines, pancreatic endocrine dysfunction, prolactin, progesterone and estrogen
Çiğ and Nazıroğlu (2015); Ghazizadeh and Nazıroğlu (2014)	Calcium overload
Aynali et al. (2013)	Melatonin lowering; sleep disruption
Othman et al. (2017a)	MicroRNA expression (brain)
Othman et al. (2017a)	Abnormal postnatal development
Çiftçi et al. (2015)	Disrupts development of teeth
Saili et al. (2015)	Cardiac changes, blood pressure disruption; erythrocyte damage
Lee et al. (2014)	Growth stimulation of adipose stem cells (role in obesity?)

Decreased spermatogenesis. Glaser (1971) also reported a total of 35 neurological/neuropsychiatric effects of non-thermal EMF exposures, including 9 central nervous system effects, 4 autonomic system effects, 17 psychological disorders, 4 behavioral changes and EEG changes. It also reported 7 types of chromosomal aberrations several of which are known to be caused by chromosomal double stranded DNA breaks, 8 types of endocrine changes, and cell death (what we now call apoptosis). Glaser (1971) also provided over 1000 different citations each reporting various types of non-thermal microwave frequency EMF effects. Consequently, the existence of 5 types of Wi-Fi effects, each supported by multiple Wi-Fi studies were already well-supported *as general non-thermal EMF effects* back in 1971, 47 years ago: effects on the testis and sperm production, neurological/neuropsychiatric effects, endocrine effects, attacks on cellular DNA and increased apoptosis/cell death.

The 146 page review published by Tolgskaya and Gordon (1973) found that in studies of histological changes in rodents, the three most sensitive organs in the body to non-thermal microwave EMFs were the nervous system (including the brain), followed closely by the heart and the testis. They also reported changes in neuroendocrine tissues and increased cell death in multiple tissues. Thus those pre-1973 rodent studies already showed that other EMFs caused 4 of the repeated, recently documented Wi-Fi effects: changes in testis structure/function, neurological effects, increased cell death (possibly via apoptosis) and endocrine effects. Findings from our longer list of EMF reviews of non-thermal effects are summarized in Table 2.

Each of the 7 Wi-Fi effects found in 2–11 studies (Table 1), have also been found to be caused by other microwave frequency EMFs, in a much larger literature (Table 2). From 10 to 16 reviews extensively document each of these seven effects as general microwave frequency effects (Table 2). These are, therefore, general effects produced by such EMFs. Each of these 7 repeatedly found Wi-Fi effects should, therefore, be considered established Wi-Fi effects. The author is not aware of any genuine Wi-Fi studies on these 7 effects that reported no statistically significant evidence of effect.

Each of these 7 is very serious: Oxidative stress has causal roles in most chronic human diseases; cellular DNA damage can cause cancer, thus producing a partial explanation for EMF cancer causation; because such DNA damage occurs in sperm cells (Atasoy et al., 2013; Avendaño the cause of each of these various other effects, as discussed below; apoptosis has central roles in neurodegenerative diseases; the neuropsychiatric effects are almost certainly caused by the impact of EMFs on brain structure which is extensively documented and, in my opinion, produces many impacts (Pall, 2016b). A recent meta-analysis shows major lowering of sperm counts and sperm quality in many countries around the world, with declines of over 50% in all advanced technology countries (Levine et al., 2017). The senior author of this study suggested that this effect alone may lead to human extinction (No authors listed, 2017). Given the major impact of EMF exposures on sperm count and quality in human and in animal studies, the pattern of evidence on male fertility is very worrying.

One thing needs to be clarified, here, however. In the two studies on calcium overload following Wi-Fi exposure, such overload was measured a substantial time period following exposure. Overload was shown to be caused, to a substantial effect, by increased TRPV1 receptor activity (Çiğ and Nazıroğlu, 2015; Ghazizadeh and Nazıroğlu, 2014). The TRPV1 receptor is known to be activated by oxidative stress. It is my view, discussed in detail below, that there is a central mechanism that acts to produce excessive intracellular calcium immediately following EMF exposure and that the oxidative stress/TRPV1 activation is secondary.

We have then, major impacts of non-thermal EMF exposures on both of the most important intercellular regulatory systems in the body, the nervous system and the endocrine systems. We have major impacts on what may be the most important intracellular regulatory system, the calcium regulatory system. And we also have non-thermal EMFs attacking the DNA of our cells, putting our biological inheritance at great risk. As living organisms, EMFs attack each of the most important functions that go to the heart of our human complexities.

Despite all of these clear and important, non-thermal effects, and the fact that there was substantial evidence for many of them already known before 1973, our current U.S. and international safety guidelines are still based on considering only thermal effects.

2. Wi-Fi and other wireless communication EMFs are pulsed, leading to larger biological impacts; These EMFs are also polarized, also producing larger effects; Dose response curves are

et al., 2012; Akdag et al., 2016; Adams et al., 2014; Liu et al., 2014; Asghari et al., 2016), such damage is highly likely to produce mutations that impact future generations; calcium overload is highly likely to be

often both non-linear and non-monotone

There are three patterns of EMF action, each of which is very important and each of which is almost universally ignored by the

Table 2
Reviews of Non-thermal Effects of Microwave Frequency EMFs Similar to Those Found in Multiple Wi-Fi Studies.

Non-thermal effects	Citations
Cellular DNA damage	Glaser (1971); Yakymenko et al. (1999); Aitken and De Iuliis (2007); Hardell and Sage (2008); Hazout et al. (2008); Phillips et al. (2009); Ruediger (2009); Makker et al. (2009); Yakymenko and Sidorik (2010); Batista Napotnik et al. (2010); Yakymenko et al. (2011); Pall (2013, 2015b); Asghari et al. (2016); Pall (2018)
Changes in testis structure, lowered sperm count/ quality	Glaser (1971); Tolgskaya and Gordon (1973); Aitken and De Iuliis (2007); Hazout et al. (2008); Desai et al. (2009); Gye and Park (2012); Nazıroğlu et al. (2013); Carpenter (2013); Adams et al. (2014); Liu et al. (2014); Houston et al. (2016); La Vignera et al. (2012); Makker et al. (2009)
Neurological/neuropsychiatric effects	Glaser (1971); Tolgskaya and Gordon (1973); Raines (1981); Lai (1994); Grigor'ev (1996); Hardell and Sage (2008); Makker et al. (2009); Khurana et al. (2010); Levitt and Lai (2010); Consales et al. (2012); Carpenter (2013); Pall (2016b); Belyaev et al. (2016); Sangün et al. (2016); Kaplan et al. (2016)
Apoptosis/cell death	Glaser (1971); Tolgskaya and Gordon (1973); Raines (1981); Yakymenko et al. (1999); Batista Napotnik et al. (2010); Yakymenko and Sidorik (2010); Pall (2013, 2016b); Asghari et al. (2016); Sangün et al. (2016)
Calcium overload	Adey (1981, 1988); Walleczek (1992); Yakymenko et al. (1999); Gye and Park (2012); Pall (2013, 2015a, 2015b, 2016a, 2016b)); Asghari et al. (2016)
Endocrine effects	Glaser (1971); Tolgskaya and Gordon (1973); Raines (1981); Hardell and Sage (2008); Gye and Park (2012); Hardell and Sage (2008); Makker et al. (2009); Pall (2015b); Sangün et al. (2016); Asghari et al. (2016)
Oxidative stress, free radical damage	Raines (1981); Houston et al. (2016); Hardell and Sage (2008); Hazout et al. (2008); Desai et al. (2009); Yakymenko and Sidorik (2010); Yakymenko et al. (2011); Consales et al. (2012); La Vignera et al. (2012); Nazıroğlu et al. (2013); Yakymenko et al. (2015); Pall (2013, 2018); Dasdag and Akdag (2016); Wang and Zhang (2017)

telecommunications industry and industry-linked organizations. The most extensively reviewed of these is that pulsed EMFs are usually much more biologically active than are non-pulsed (also known as continuous wave) EMFs of identical frequency and similar average intensity (Osipov, 1965; Pollack and Healer, 1967; Creighton et al., 1987; Grigor'ev, 1996; Belyaev, 2005, 2015; Markov, 2007; Van Boxem et al., 2014; Pall, 2015b; Panagopoulos et al., 2015b). This pattern of action is particularly important because all wireless communication devices, including Wi-Fi (Panagopoulos et al., 2015b; Maret, 2015) communicate via pulsations and are likely to be particularly dangerous as consequence of this. Panagopoulos et al., 2015b have argued that the more pulsed they are, the more damaging EMFs will be and while this may still be questioned, it may well be a roughly applicable generalization.

It is also true that artificial EMFs are polarized and this makes artificial EMFs particularly dangerous (Belyaev, 2005, 2015; Panagopoulos et al., 2015a). Polarized EMFs put much larger forces of electrically charged chemical groups than do non-polarized EMFs (Panagopoulos et al., 2015a), an observation that is relevant to the main mechanism of EMF action in living cells discussed below.

It has often been found that there are windows of exposure where specific intensity ranges produce maximum biological effects, which drop off going to both lower or higher intensities (Belyaev, 2005, 2015; Pall, 2015b). It can be seen from this that dose-response curves are often both non-linear and non-monotone whereas industry linked groups often assume a linear and therefore monotone dose-response curve.

3. EMF effects are often cumulative and irreversible

One question that has been raised about the effects of these low-intensity EMFs producing biological effects is are they cumulative? I am aware of three different types of evidence for cumulative effects. Three of the human occupational exposure studies from the 1970's reviewed in Raines (1981), showed that effects increased substantially with increasing time of exposure to a particular type and intensity of EMF.

The impacts of such EMFs on animal brains were reviewed in Tolgskaya and Gordon (1973) and discussed in Pall (2016b). Initially exposures over period of 1–2 months produced relatively modest changes in structure of the brain and the neurons and when exposures ceased, most of the structural changes disappeared – that is the changes ground at two locations each with somewhat different exposures within an antenna park. The exposure levels at both sites were well within safety guidelines, so if the safety guidelines have any biological relevance, there should have been no apparent effects. It takes about 30 days for mice to go through gestation. At the higher level exposure, the pairs produced one litter of lower than normal size, and a second litter with lowered numbers of progeny; after that they were completely sterile or had extremely low fertility (Magras and Xenos, 1997). At the other site, the mating pairs produced four litters, with decreasing numbers of progeny over time followed by complete sterility. In both groups, the mating and possible subsequent gestatation for the fifth possible litter were performed under conditions of no EMF exposure, but the fertility effects were not reversed; therefore fertility effects may become irreversible, suggesting a similar pattern to the brain related effects of EMFs. It should be noted that Özorak el al (2013) showed that Wi-Fi exposure impacted animal reproduction and that (Atasoy et al., 2013; Shokri et al., 2015; Dasdag et al., 2015; Avendaño et al., 2012; Yildiring et al., 2015; Oni et al., 2011; Akdag et al., 2016) suggest this as well from the Wi-Fi impacts on testis structure and sperm production.

Mutation accumulation produced by cellular DNA damage is likely to be both cumulative and irreversible, as well, because later mutations are highly unlikely to reverse previously occurring mutations.

We have therefore reason to think that such effects as brain damage to animal brains, neuropsychiatric effects in humans, reproductive dysfunction in mice and mutational effects, are each cumulative. Those same effects may be completely or largely irreversible. One thing that this should tell us is that the short-term Wi-Fi studies shown in Table 1 may *greatly underestimate* the damage Wi-Fi may do over much longer time periods. Given the fact that Wi-Fi has been placed in most schools, hotels, restaurants, coffee shops, commercial aircraft and airports as well as in many homes and that Wi-Fi hot spots are becoming increasingly common in cities around the world, we should expect massive cumulative Wi-Fi effects in many people. A second tentative inference is that false assurances of safety on the part of industry are likely to lead to much more severe effects on people exposed to Wi-Fi or other EMFs; rather than leading them to protect themselves or their children by avoiding exposures or demanding that others stop non-voluntary exposures, they are likely to avoid protective changes or be prevented from doing such protective changes. A third inference is that these effects may be among the more difficult ones for us to attribute to EMF exposure. We are much more aware of effects that occur rapidly than those that take months or years before they become readily ap-

were largely reversible. However more months of exposure produced much more severe impacts on brain and neuronal structure and these were irreversible (Tolgskaya and Gordon, 1973; Pall, 2016b).

Magras and Xenos (1997) put pairs of young mice into cages on the

than those that take months or years before they become readily apparent.

4. Wi-Fi and other EMFs may be particularly damaging to young people

Most arguments that have been made that microwave frequency EMFs may be much more damaging to young children have centered on the much smaller skulls and skull thickness in young children, increasing the exposure of their brains to EMFs (Gandhi and Kang, 2001; Gandhi et al., 2012). However there are other arguments to be made. EMFs have been shown to be particularly active in producing effects on embryonic stem cells (Lee et al., 2014; Belyaev et al., 2009; Markovà et al., 2010; Czyz et al., 2004; Xu et al., 2016; Bhargav et al., 2015; Odaci et al., 2008; Uchugonova et al., 2008; Wang et al., 2015; Teven et al., 2012). Because such stem cells occur at much higher cell densities in children, with stem cell densities the highest in the fetus and decreasing with increasing age (Belyaev et al., 2009; Markovà et al., 2010), impacts on young children are likely to be much higher than in adults. The decreased DNA repair and increased DNA damage following EMF exposure strongly suggest that young children may be increasingly susceptible to cancer following such exposures (Belyaev et al., 2009; Markovà et al., 2010; Czyz et al., 2004). EMF action on stem cells may also cause young children to be particularly susceptible to disruption of brain development (Xu et al., 2016; Bhargav et al., 2015), something that may be relevant to autism causation. These are all very problematic issues and we cannot rule out the possibility that there are other problematic issues as well. Redmayne and Johansson (2015) reviewed the literature showing that there are age-related effects, such that young people are more sensitive to EMF effects. It follows from these various findings that the placement of Wi-Fi into schools around the country may well be a high level threat to the health of our children as well being a threat to teachers and any very sensitive fetuses teachers may be carrying, as well.

5. How do EMF exposures lead to non-thermal health impacts?

The author found the answer to this question in the already published scientific literature (Pall, 2013). That study showed that in 24 different studies [there are now a total of 26 Pall (2015b)], effects of low-intensity EMFs, including microwave frequency and also extremely low frequency EMFs, static electrical fields and static magnetic fields could be blocked by calcium channel blockers, drugs that are specific for blocking voltage-gated calcium channels (VGCCs). There were 5 different types of calcium channel blockers used in these studies, each thought to be highly specific, each structurally distinct and each binding to a different site on the VGCCs. In studies where multiple effects were studied, all studied effects were blocked or greatly lowered by calcium channel blockers. These studies show that EMFs produce diverse non-thermal effects via VGCC activation Pall (2013, 2014, 2015a, 2015b, 2016a, 2016b)) in many human and animal cells. In plant cells, EMFs activate somewhat similar calcium channels and produce somewhat similar effects on oxidative stress, cellular DNA damage and calcium signaling (Pall, 2016a). Furthermore, many different effects shown to be produced in repeated studies by EMF exposures, including the effects discussed above, can be produced by downstream effects of VGCC activation, via increased [Ca2+]i, as discussed in detail below.

Before leaving this issue, it is important to discuss why the VGCCs are so sensitive to activation by these low-intensity EMFs. The VGCCs each have a voltage sensor which is made up of 4 alpha helixes in the plasma membrane, with each such helix having 5 positive charges on it, for a total of 20 positive charges (Pall, 2015b). These voltage sensor helixes are each called S4 helixes because each is the fourth helix in a distinct multi-helix domain. Each of these voltage sensor charges is single charge. 2. Because these charges are within the lipid bilayer section of the membrane where the dielectric constant is about 1/120th of the dielectric constant of the aqueous parts of the cell, the law of physics called Coulomb's law, predicts that the forces on those charges will be approximately 120 times higher than the forces on charges in the aqueous parts of the cell. 3. Because the plasma membrane has a high electrical resistance whereas the aqueous parts of the cell are highly conductive, the electrical gradient across the plasma membrane is estimated to be concentrated about 3000-fold. The combination of these effects means that comparing the forces on the voltage sensor with the forces on singly charged groups in the aqueous parts of the cell, the forces on the voltage sensor are approximately $20 \times 120 \times 3000 = 7.2$ million times higher (Pall, 2015b). The physics predicts, therefore, extraordinarily strong forces activating the VGCCs via the voltage sensor. It follows that the biology tells us that the VGCCs are the main target of the EMFs and the physics tells us why they are the main target. Thus the physics and biology are pointing in the same direction.

There are also additional findings pointing to the voltage sensor as the direct target of the EMFs. In addition to the VGCCs, there are also voltage-gated sodium, potassium and chloride channels, with each of these having a voltage sensor similar to those found in the VGCCs. Lu et al. (2015) reported that voltage gated sodium channels, in addition to the VGCCs were activated by EMFs. Tabor et al. (2014) found that Mauthner cells, specialized neurons with special roles in triggering rapid escape mechanisms in fish, were almost instantaneously activated by electrical pulses, which acted via voltage-gated sodium channel activation to subsequently produce large [Ca2+]i increases. Zhang et al. (2016) reported that in addition to the VGCCs, potassium and chloride channels were each activated by EMFs, although these other voltage-gated ion channels had relatively modest roles compared with the VGCCs in producing biological effects. Each of these three studies, the Lu et al. (2015) study, the Tabor et al. (2014) study and the Zhang et al. (2016) study used specific blockers for these other voltage-gated ion channels to determine their roles. The Tabor et al. (2014) study also used genetic probing to determine the role of the voltage-gated sodium channels. Lu et al. (2015) also used whole cell patch clamp measurements to measure the rapid influx of both sodium and calcium into the cell via the voltage-gated channels following EMF exposure. Sodium influx, particularly in electrically active cells, act in the normal physiology to depolarize the plasma membrane, leading to VGCC activation such that the voltage-gated sodium channels may act primarily via indirect activation of the VGCCs. In summary then, we have evidence that in animal including human cells, seven distinct classes of voltage-gated ion channels are each activated by EMF exposures: From the Pall (2013) review, four classes of voltage-gated ion channels were shown from calcium channel blocker studies, to be activated by EMFs, L-type, T-type, N-type and P/Q –type VGCCs. In this paragraph we have evidence that three other channels are also activated, voltage-gated sodium channels, voltage-gated potassium channels and voltage-gated chloride channels. Furthermore the plant studies strongly suggest that the so called TPC channels, which contain a similar voltage sensor, are activated in plants allowing calcium influx into plants to produce similar EMF-induced responses (Pall, 2016a). One can put those observations together with the powerful findings from the physics, that the electrical forces on the voltage-sensor are stunningly strong, something like 7.2 million times stronger than the forces on the singly charged groups in the aqueous phases of the cell. Now you have a stunningly powerful argument that the voltage sensor is the predominant direct target of the EMFs.

There is one additional finding that should be discussed here. In a study published by Pilla (2012), it was found that pulsed EMFs pro-

within the lipid bilayer part of the plasma membrane. The electrical forces on the voltage sensor are very high for three distinct reasons (Pall, 2015b, 2015a, 2016a). 1. The 20 charges on the voltage sensor make the forces on voltage sensor 20 times higher than the forces on a

duced an "instantaneous" increase in calcium/calmodulin-dependent nitric oxide synthesis in cells in culture. What Pilla (2012) showed was that following EMF exposure, the cells in culture, must have produced a large increase in [Ca2 +]i, this in turn produced a large increase in

nitric oxide synthesis, the nitric oxide diffused out of the cells and out of the aqueous medium above the cells into the gas phase, where the nitric oxide was detected by a nitric oxide electrode. This entire sequence occurred in less than 5 s. This eliminates almost any conceivable indirect effect, except possibly via plasma membrane depolarization. Therefore that the pulsed EMFs are acting directly on the voltage sensors of the VGCCs and possibly the voltage-gated sodium channels, to produce the [Ca2 +]i increase.

Why is it that the VGCCs, acting via calcium influx, seem to be much more important in producing EMF effects than are the other voltage-gated ion channels? Probably for three reasons: 1. Ca^{2+} ions under resting conditions in cells have about a 10,000-fold concentration gradient driving them into the cell, and over a million-fold electrochemical gradient also driving them into the cell. Because of this, one can have huge calcium influxes upon channel activation. 2. [Ca2 +]i produces many important regulatory effects, such that over activation of those effects can have very large pathophysiological consequences. 3. Sustained elevation of [Ca2+]i produces major cell damage.

6. How can the Wi-Fi effects be produced by EMF triggered VGCC activation?

Can the various effects produced by Wi-Fi and by other microwave frequency EMFs be produced by the downstream effects of VGCC activation? In order to determine that, one needs to consider the various downstream effects of VGCC activation, summarized in Fig. 1 and how these are likely to produce each of the effects of Wi-Fi and other microwave frequency EMFs. Let's consider Fig. 1.

As shown in the top left section of Fig. 1, microwave and lower frequency EMFs act via VGCC activation to produce increases in intracellular calcium [Ca2+]i. All of the downsteam effects of VGCC activation considered in Fig. 1 are produced by elevated (often excessive) [Ca2+]i.

Just to the right of [Ca2+]i in Fig. 1, you will see that elevated [Ca2+]i produced increases in nitric oxide (NO) synthesis. This is because two of the three types of enzymes producing NO are calcium-dependent. There is an NO signaling pathway that goes through increased cGMP and increased protein kinase G activity. Protein kinase G can act by raising the activity of the transcriptional regulatory factor, Nrf2, to produce the therapeutic effects produced by EMF exposures (Pilla, 2013; Pall, 2014; Pall and Levine, 2015).

High levels of NO can bind to heme groups on cytochromes (uppermost section, Fig. 1) inhibiting cytochrome oxidase, the terminal oxidase in the mitochondria, inhibiting ATP synthesis. NO can also inhibit cytochrome P450s involved in steroid hormone synthesis, lowering levels of estrogen, progesterone and testosterone (sex hormones).

The main pathophysiological effects of EMF exposures are produced via excessive calcium signaling (lower left) and the peroxynitrite pathway (lower right). Peroxynitrite levels are elevated because both NO and superoxide are elevated by increased [Ca2+]i with NO and superoxide reacting with each other to form peroxynitrite. Peroxynitrite and its CO_2 adduct, can break down to produce reactive free radicals, hydroxyl radical, carbonate radical and NO_2 radical which produce oxidative stress. These various oxidants act to produce greatly elevated NF-kappaB activity, leading to inflammation. All of this biochemistry and physiology is well-accepted and widely known with a single exception: The role of protein kinase G in raising Nrf2 has only recently been reviewed (Pall and Levine, 2015).

The ways in which these mechanisms can produce each of the seven effects produced by Wi-Fi, as well as other microwave frequency EMFs, are described in Table 3.

It can be seen from Table 3, that there are plausible mechanisms by which each of those seven effects can be produced by VGCC activation via known pathways. Given the complexities of biology, the mechanisms described in Table 3 may, in some cases, be over simplified.

There is one other finding, not related to the Wi-Fi findings, that is included in Table 3. A question that was raised in review of the paper was whether the heat shock stress elevation found following EMF exposure in many studies, could be produced by VGCC activation. As you can see from Table 3, it can be.

7. Other proposed biophysical mechanisms

One question that can be asked is how the VGCC activation mechanism compares with other biophysical models of non-thermal EMF effects. Belyaev (2015) has discussed a number of what he describes as biophysical models which are, therefore considered here. These models are basically theoretical models of how the weak electrical forces of the EMFs can interact with biologically plausible structures to produce non-thermal effects.

The first of these Belyaev considers is Fröhlich's theory. This is where there are “coherent longitudinal vibrations of electrically polar structures.” The mechanism of Fröhlich's theory will not be considered here (the reader is referred to Belyaev, 2015). The author considers this to be a plausible mechanism for possible production of some non-thermal EMF effects. However, there are no specific testable predictions made by the theory that suggest how it could be tested, given the fact that there may be multiple possible targets of the EMFs according to

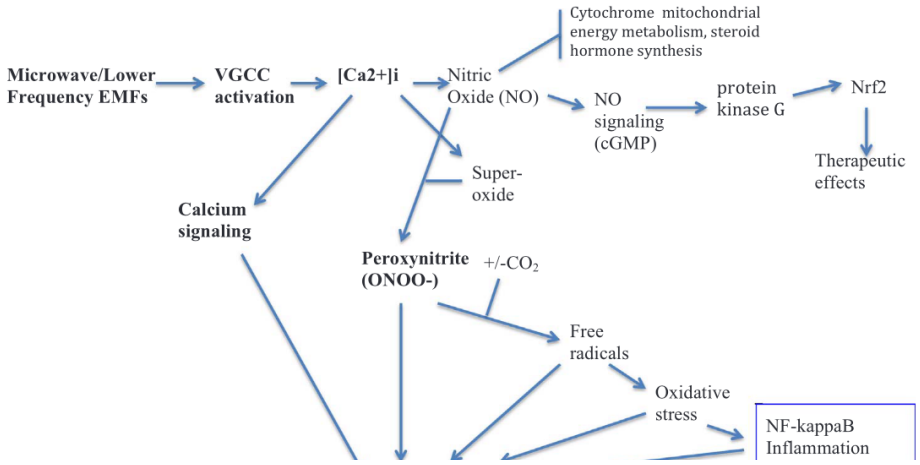

Fig. 1. Various pathways of action by which EMF VGCC activation can produce effects produced by EMF exposure (modified, with permission from Pall, 2015b).

409

Table 3
How Eight Established Effects of Wi-Fi and Other EMFs Can Be Produced by VGCC Activation.

EMF effect	Probable mechanism(s)
Oxidative stress	Produced by elevated levels of peroxynitrite and the free radical breakdown products of peroxynitrite and its CO_2 adduct. Four studies of EMF exposure, cited in Pall (2013) showed that oxidative stress following exposure was associated with major elevation of 3-nitrotyrosine, a marker of peroxynitrite, thus confirming this interpretation. Two other studies each found 3-nitrotyrosine elevation, both following 35 GHz exposures (Sypniewska et al. (2010); Kalns et al., 2000).
Lowered male/female fertility, elevated spontaneous abortion, lowered libido	Both the lowered male fertility and lowered female fertility are associated with and presumably caused by the oxidative stress in the male and female reproductive organs. Spontaneous abortion is often caused by chromosomal mutations, so the germ line mutations may have a causal role. Lowered libido may be caused by lowered estrogen, progesterone and testosterone levels. It seems likely that these explanations may be greatly oversimplified. One mechanism that may be important in lowered fertility is that VGCC activation and consequent high {Ca2+]i levels is known to have a key role in avoiding polyspermy. Consquently, if this if triggered before any fertilization of an egg has occurred, it may prevent any sperm from fertilizing and egg.
Neurological/ neuropsychiatric effects	Of all cells in the body, the neurons have the highest densities of VGCCs, due in part to the VGCC role and [Ca2+]i role in the release of every neurotransmitter in the nervous system. Calcium signaling regulates synaptic structure and function in 5 different ways, each likely to be involved here. Oxidative stress and apoptosis are both thought to have important roles. Lowered sleep and increased fatigue are likely to involve lowered nocturnal melatonin and increased nocturnal norepinephrine.
Apoptosis	Apoptosis can be produced by excessive Ca2+ levels in the mitochondria and by double strand breaks in cellular DNA; it seems likely that both are involved following EMF exposure. A third mechanism for triggering apopotosis, endoplasmic reticulum stress (see bottom row in this Table), may also be involved.
Cellular DNA damage	Cellular DNA damage is produced by the free radical breakdown products of peroxynitrite directly attacking the DNA [see Pall (2018) for discussion].
Changes in non-steroid hormone levels	The release of non-steroid hormones is produced by VGCC activation and [Ca2+]i elevation. The immediate effects of EMF exposures is to increase hormone release and to raise, therefore, hormone levels. However many hormone systems become "exhausted" as a consequence of chronic EMF exposures. The mechanism of exhaustion is still uncertain, but it may involve oxidative stress and inflammation.
Lowered steroid hormone	Steroid hormones are synthesized through the action of cytochrome P450 enzymes; activity of these hormones is inhibited by binding of high levels of nitric oxide (NO) leading to lowered hormone synthesis.
Calcium overload	Produced by excessive activity of the VGCCs; secondary calcium overload is produced by oxidative stress activation of TRPV1, TRPM2 and possibly some other TRP receptors, opening the calcium channel of these receptors.
Heat shock protein induction	There is a large literature showing that excessive [Ca2+]i induces very large increases in heat shock proteins. This is thought to be produced by complex calcium signaling changes involving the endoplasmic reticulum, mitochondria and the cytosol and also involving excessive [Ca2+]i producing increasing protein misfolding (Garbuz, 2017; Park et al., 2014; Krebs et al., 2011). It should be noted that some calcium is essential for proper protein folding in the endoplasmic reticulum such that only excessive calcium leads to misfolding and consequent endoplasmic reticulum stress.

Fröhlich's theory.

A second possible mechanism involves the spin state of radical pairs. When radical pairs are generated from the breakdown of a non-radical molecule, these radical pairs often react back with each other to form another non-radical molecule, not necessarily identical to the original non-radical. What is postulated by this theory is that EMFs can interact with one or both radicals, changing their spin state and greatly lowering their ability to react back with each other, thus generating increased free radicals and therefore increased oxidative stress. The potential strong point of this theory is that it provides an explanation for the oxidative stress found following EMF exposure. However, as noted under oxidative stress in Table 3, there are 6 studies where oxidative stress following EMF exposure was associated with very high levels of 3-nitrotyrosine, a specific marker of peroxynitrite elevation. These studies argue, therefore, that oxidative stress following EMF exposure is produced by peroxynitrite elevation and is not primarily produced by this radical pair mechanism. It follows from this that the proposed radical pair mechanism cannot even explain the properties of oxidative stress production, let alone the various consequences of non-thermal EMF exposure that do not involve oxidative stress. Does that mean that the radical pair mechanism has no possible role in producing non-thermal EMF effects? No, but it does argue there is no evidence for any such role.

A third mechanism discussed in Belyaev (2015) is the electrosoliton theory proposed by Brizhik and colleagues, involving a "self reinforcing solitary wave packet." Brizhik and her colleagues discussed this in the substantial similarity to what is thought to occur in the activation of the voltage sensor, that is discussed above. There we have four alpha helixes, each designated an S4 helix and with each S4 helix having 5 positive charges, with the 4 S4 helixes together making up the voltage sensor. Most of those positive charges are 3 amino acid residues apart from each other, such that the closest charged residues stick out from the helix pretty much on the same side of the helix. Three of those positive charges are electrostatically attracted to negative residues on other helixes thought to be in fixed positions. What is thought to happen in activation is that there a ratcheting of the S4 helixes toward the extracellular space, ratcheting such that the negative charges are now bound to a positive charge 3 residues away from the one that was previously bound. The ratcheting also produces some turning of the S4 helix. This needs to occur several times on each of the four S4 helixes to open the channel and allow calcium ions to flow. While I don't completely understand the Brizhik electrosoliton model, it may well be relevant to our understanding the VGCC activation, because the mechanism of the voltage sensor is similar to what Brizhik and her colleagues propose to occur in the electrosoliton model. Both the electrosoliton model and the voltage sensor activation mechanism involve both charge movements and ratcheting. In order to test these biophysical models one needs to have a specific mechanism where it may apply and where such tests can be done. In the case of the voltage sensor of the VGCCs, these tests have already been done.

These models are basically theoretical models of how the weak electrical forces of the EMFs can interact with biologically plausible

context of reaching a threshold minimum energy state where both charged molecules and the EMF is in a coherent state, such that charge movement can ratchet from one state to another. This concept shows structures to produce non-thermal effects. Their theoretical support is their strong point. They are weak, however, in providing any compelling evidence that they have causal roles in producing non-thermal

changes in cells in culture or in whole animal (or human) studies. They are also weak because they do not provide stated explanations for the range of EMF effects that have been documented.

Belyaev (2015) discusses microwave hearing in this context. He discusses the findings showing that people can hear microwave fields that are pulsed, including pulsed low intensity EMFs. While there is no doubt that these are very interesting observations on what are clearly non-thermal effects, they do not provide a biophysical model explaining how microwave hearing may occur. It is important, therefore to ask whether such microwave hearing could be caused by VGCC activation. It has been shown that hearing involves the activation of the VGCCs (Joiner and Lee, 2015). Furthermore, various otolaryngological conditions, including tinnitus, involve excessive VGCC activity, such that the calcium channel blocker, nimodipine is useful in their treatment (Monzani et al., 2015). These findings tells us that microwave hearing may be produced by VGCC activation. Consequently, microwave hearing may be interpreted as providing further support for the VGCC mechanism.

Following microwave hearing, Dr. Belyaev (2015) discusses plasma membrane and ion models. Here the VGCC mechanisms fit into the scheme, as do the other voltage-gated ion channels and the plant TPC channels, all discussed above as being activated by their voltage sensor following EMF exposures.

Finally, Dr. Belyaev (2015) discusses possible direct effects of EMFs on DNA, possibly leading to changes in chromatin structure and/or nuclear structure. There is a literature showing that aqueous solutions of DNA absorb microwave EMFs much more efficiently than do identical solutions not containing DNA. This clearly shows that DNA has a high absorbance of the EMFs, Furthermore, there are studies showing such dissolved DNA, when it absorbs such EMFs, undergoes structural changes as measured by biophysical techniques. All of this suggests that DNA is a plausible potential target for the EMFs. The problem is what are the predicted effects of such changes in DNA structure in living cells and organisms? Dr. Belyaev spends almost a page and a half in his paper discussing various possible models of interactions of DNA or of chromatin with EMFs. But again, how do we test any of these in living cells to demonstrate a role of such DNA or chromatin changes in producing any specific or general biological effects? Given the extraordinary complexity of living cells and organisms, there are only two powerful ways of demonstrating causal roles in such living cells and organisms. These are to use genetics or to use specific pharmacological agents. The extraordinary power of each of these approaches comes from the fact that these approaches allow researchers to vary one variable at a time out of the thousands of interacting variables in a living cell, allowing us to ask does that specific variable have a causal role in determining a specific response. But these two approaches can be used when specific proteins have specific roles, not when you are looking at the role of DNA structural changes, Fröhlich's theory, radical pair mechanisms or electrosoliton models. Fortunately the VGCC mechanism does allow this approach by studying various classes of calcium channel blockers, so here we do have hard data on widespread causal roles of VGCC activation in producing EMF effects.

8. Two other models for producing non-thermal effects

With the possible exception of the electrosoliton model, the author does not find any of the models discussed by Dr. Belyaev (2015) to have substantial evidence for roles in producing EMF effects. There are two other models which may be more compelling, each of which either produces increased [Ca2+]i.

Six studies have supported the view that calcium cyclotron resonance, has a role in producing biological effects produced by *certain* which produces the calcium cyclotron resonance and in three studies, these frequencies were shown to produce increases in [Ca2+]i levels. In the De Carlo et al. (2012) study, the calcium channel blocker nifedipine was shown to greatly lower the apparent calcium cyclotron resonance effect. This finding strongly suggests that the calcium cyclotron resonance can feed Ca^{2+} ions into the VGCCs, thus increasing the flow of Ca^{2+} ions through the VGCCs into the cell following EMF exposure. The frequencies studied here for cyclotron resonance, one was close to 7 Hz and the other was close to 50 Hz, are both in the extremely low frequency range and consequently are not relevant to microwave frequency effects. The finding that only very specific calcium cyclotron resonance frequencies produce these effects is the main evidence for this mechanism.

It is now well established that there is a magnetoreception mechanism found in many animals that can detect and respond to the very low intensity geomagnetic field. This has been most studied in bees and in birds, both of whom use it for navigation. This has been suggested to involve tiny particles of magnetite which occur in bacterial, animal and plant cells, including human cells. Kirschvink (1992) first proposed a model of how such a mechanism might act. He proposed that magnetite particles may be tethered through a microtubule and/or microfilament or perhaps other fibers to a mechanosensitive channel, such that tiny magnetic forces could open the mechanosensitive channels, allowing cation flow into the cells. It is still uncertain what mechanosensitive channel or channels might be involved, but most of the candidates are channels that allow both sodium and calcium to flow into cells. Hsu et al. (2007) suggested that such magnetite particles were linked in honeybees to an undefined calcium channel, such that magnetic field exposure produces increases in [Ca2+]i. The worm Caenorhabditis elegans had been shown to have a geomagnetic orientation system. Vidal-Gadea et al. (2015) found that certain specific neurons in C. elegans which may be geomagnetic sensory neurons, very low intensity geomagnetic fields could produce increases in [Ca2+]i in those specific neurons, even when they had no synaptic inputs, suggesting that these neurons themselves acted as geomagnetic sensors.

Cadiou and McNaughton (2010) reviewed the literature on a magnetite-based magnetoreception system in birds and its role in avian migration. They also reviewed findings on neurons found in the trigeminal nerve of birds, where magnetic fields as low as 200 nT can activate specific neurons. Trains of action potentials are produced by magnetic fields, plateauing in the region of 20–100 mT. Latency in a study presented by Cadiou and McNaughton (2010) was about 4 s, but other studies have reported latencies of about 2.5 s. Therefore these are rapid effects. Cadiou and McNaughton (2010) also discuss possible roles mechanosensitive channels, including a model similar to that proposed by Kirschvink (1992) and also three other models, each involving different ways of coupling forces on magnetite to opening of a channel. Magnetoreception has also been reported to occur in a mammal, the mole-rat (Wegner et al., 2006). There are also studies of magnetic compass orientation in salmonids, newts, sea turtles and other rodents. There is evidence in Drosophila, that a magnetic structure attached to cryptochrome is involved in magnetoreception, as opposed to magnetite.

The two mechanisms described in this section have minor roles, only acting, as far as we can tell, in very specific situations. The calcium cyclotron resonance mechanism only acts with a few specific frequencies in the extremely low frequency range. The magnetoreception mechanism only acts, as far as one can tell, on detecting the weak geomagnetic fields and only acts, as far as one can tell, in certain specific neurons. It is possible that this view may change with regard to the magnetoreception mechanism but what is clear is that the VGCC mechanism is vastly more important than either of these mechanisms,

specific frequencies which can interact with Ca^{2+} ions to produce a cyclotron-like resonance (Foletti et al., 2010; Gaetani et al., 2009; De Carlo et al., 2012; Lisi et al., 2008; Pazur and Rassadina, 2009; Pazur et al., 2006). In each case, the effects involved a very specific frequency acting in diverse cell types and acting to provide responses to a very wide frequency range and even to static electrical fields and static magnetic fields. Because static magnetic fields only place forces on moving electric charges, this produced a puzzle on how they can

activate the VGCCs. Pall (2013) suggested that the solution to that puzzle is that the plasma membrane of animal cells is often moving, such that the charges in the voltage sensor are also moving and can, therefore, have forces placed on them by the static magnetic fields. These static magnetic fields, activating the VGCCs can be relative low intensity but probably must be much higher intensity than the extraordinarily weak geomagnetic fields. The reader is referred to Lu et al. (2015) for empirical information from an important static magnetic field study, where those static magnetic fields activate both VGCCs and voltage-gated sodium channels.

9. Foster and Moulder on Wi-Fi

The Foster and Moulder (2013) paper argues that there are no and cannot be any health effects of Wi- Fi. The first 7½ pages of the paper are, however, largely irrelevant to that issue. These pages discuss such issues as predicted peak power output, incident power density and the FCC and international safety guidelines. They also discuss specific absorption rate (SAR) values, a measure of heating. Because it is now established, as discussed above that thermal effects are not the relevant mechanism of non-thermal effects and that VGCC activation is the main mechanism of such effects, this whole section is irrelevant. Foster and Moulder (2013) discuss the issue of biological effects, praising 7 studies listed in table 4 of their paper as having "well-characterized exposure systems" of well defined SARS values, reporting that there were no effects in the rats or mice in those 7 studies. Those 7 studies are Laudisi et al. (2012), Sambucci et al. (2010), Aït-Aïssa et al. (2010, 2012, 2013) and Poulletier de Gannes et al. (2012, 2013). The first two studies come from one research group and the other five from another, albeit with some shared personnel.

Six or those seven studies (Sambucci et al., 2010; Aït-Aïssa et al., 2010, 2012, 2013; Poulletier de Gannes et al., 2012, 2013) used an exposure system described by Wu et al. (2009) that is important here and that was claimed to produce a near uniform exposure. Laudisi et al. (2012) used a somewhat similar exposure system of Ardoino et al. (2005), albeit another one that is also claimed to produce near uniform exposures. The important features here of the Wu et al. (2009) exposure system need to be examined in the light of the fact that, as discussed above, artificial EMFs are polarized with the polarization producing much larger biological effects than natural non-polarized EMFs (Belyaev, 2005, 2015; Panagopoulos et al., 2015a). The probable important feature of these polarized EMFs is that they put much larger forces on electrically charged groups (Panagopoulos et al., 2015a); since such forces are central to VGCC activation via the voltage sensor, as discussed above, they are likely to be central to the production of most biological effects. Let's examine Wu et al. (2009) with that issue in mind. It uses a large chamber surrounded by 1 mm aluminum mesh wire mesh to provide reflections of the EMFs. The chamber in which animals are exposed on a platform at its center, is also surrounded by antennae in all 6 directions (up, down, all four horizontal directions) such that each antenna is broadcasting with one polarization is opposed (at 180°) by another broadcasting with the 180° opposite polarization, as well as by four other antennae, broadcasting with 90° different polarization in each of the four possible directions. This produces a field that is more like a non-polarized EMF rather than the usual polarized artificial EMF. This move toward non-polarization is further exacerbated by the aluminum wire reverberation system whose reflections will generate vast numbers of reflections of different polarity, like a non-polarized EMF. The consequences of this is that the structure of this exposure system is clearly very different from that seen in Wi-Fi or any other artificially produced EMF that we may be exposed to, with biological effects produced via electrical forces being vastly less. Conexposure systems. Because of the many reverberations occurring, the path lengths of different photons reaching a specific point in the exposed tissue, will often be quite different from each other, such that the phase of the EMFs produced will also be quite different from each other. This leads to the possibility of destructive interference and thus a second mechanism which is predicted to lead to substantial decreases in the intensity of the exposures. Because exposures are usually predicted by groups using such exposure chambers without considering such destructive interference, rather than being measured, the actual exposures may be substantially lower than are the predicted exposures. Both the polarization effect and the possible difference between predicted exposure and actual exposure were considered in an earlier study.

Vian et al. (2006), using a different reverberation exposure chamber, discussed in Fig. 1 of that paper, how the various reverberations lead to the initial polarized EMF being converted to a non-polarized or at least, less polarized EMF. They also on p. 69 if that paper compared the predicted with the measured amplitude and found that the measured amplitude was only 78% of the predicted amplitude. These findings suggest that both of the lowered polarization and destructive interference discussed in the previous two paragraphs can have substantial roles in lowering biological responses produced when using such reverberation exposure chambers.

Laudisi et al. (2012) used a different exposure system, that of Ardoino et al. (2005) where the vast majority of the exposure is produced from reflections off a long cylindrical surface in a TEM cell, where the curvature of the cylinder will also produce a largely non-polarized EMF and different reverberation paths and consequent destructive interference, may both be expected to occur. Consequently the predicted low biological activity of EMFs produced by the Wu et al. (2009) system may be expected to also occur from this TEM exposure system Ardoino et al. (2005). It is not possible to study biological effects of EMFs from Wi-Fi, cell phones or any other important exposures using such exposure systems because of the polarization changes they produce from the original polarized EMFs and because of destructive interference.

Let's now shift to the issue of the important role of pulsations in producing biological effects and ask whether the EMFs fed into the antennae have pulsation patterns similar or different from genuine Wi-Fi. Poulletier de Gannes et al. (2012) used a non-pulsed (continuous wave) as did Wu et al. (2009), an EMF which will have, therefore, much lower biological effects that genuine Wi-Fi with its myriad of pulsations (Maret, 2015). The other 6 studies (Laudisi et al., 2012; Sambucci et al., 2010; Aït-Aïssa et al., 2010, 2012, 2013; Poulletier de Gannes et al., 2013) used computers with Wi-Fi cards. Such Wi-Fi cards are designed to communicate with genuine Wi-Fi antennae, but are used here to communicate with each other, using two such computers to generate "Wi-Fi". How the EMFs so generated compare with the pulsations of genuine Wi-Fi is a complete mystery and none of these papers provide any information to allow the reader to make such a comparison. It follows that these studies (Laudisi et al., 2012; Sambucci et al., 2010; Aït-Aïssa et al., 2010, 2012, 2013; Poulletier de Gannes et al., 2013) are not studying genuine Wi-Fi, even before the effects of the reverberation chamber and the reader is left with no evidence to compare these original EMFs with genuine Wi-Fi. In summary, then none of the EMFs used in these studies are genuine Wi-Fi, with them differing from genuine Wi-Fi in three different ways: the antenna locations produce a substantial difference from genuine Wi-Fi regarding EMF polarization and this is further exacerbated by the effects of the aluminum mesh reverberation producing further lowering of any polarization; differences in path lengths of different photons produce substantial destructive interference; the initial EMF fed into the antennae differs

sequently this exposure system is not only inherently different from genuine Wi-Fi, it is predicted to be inherently less active than genuine Wi-Fi, regardless of what EMFs are being fed into the 6 antennae.

There is a second type of consequence of using such reverberation substantially from genuine Wi-Fi, with the main concern here being due to the issue of pulsation patterns and biological effects.

Let's shift now to the claim made by Foster and Moulder (2013) that there were no effects found in any of these 7 studies. Rothman et al.,

Modern Epidemiology, 3rd Edition is a highly respected source of information, cited over 18,500 times according to the Google Scholar database. It states (p. 151, bottom) that: "A common misinterpretation of significance tests is that there no difference between two observed groups because the null test is not statistically significant, in that P is greater that the cutoff for declaring statistical significance (again, usually .05). This interpretation confuses a descriptive issue (whether two observed groups differ) with an inference about the superpopulation. The significance test refers only to the superpopulation, not the observed groups. To say that the difference is not statistically significant means only that one cannot reject the null hypothesis that the superpopulation groups are the same; it does not imply that the two groups are the same." It follows that the claim of "no effect" that Foster and Moulder (2013) make about each of these 7 studies in Table 4 of their paper is false because one can never legitimately make such a claim; one can at most claim that there were no statistically significant differences.

However there are other reasons to reject those claims that need to be considered for each of these 7 studies. Each of these 7 studies fails to provide raw numerical data, the lack of which is problematic, given the other flaws that follow. **1).** Laudisi et al. (2012) finds in Table 2, that two T cell populations are statistically significantly different in pre-natally exposed mice vs sham controls: DP and CD4SP cells are significantly affected by exposure in mice at 26 weeks after birth; CD4SP cells are affected in female mice at 5 weeks after birth ($P < .02$ in each case). Furthermore in each of the measurements in Laudisi et al. (2012), only 11 or 12 mice were studied, tiny numbers. It follows that claims in Foster and Moulder (2013) that there were no effects are false or misleading for 3 distinct reasons: You can never make such claims even in large studies; there were 3 comparisons each of which showed statistically significant effects; this study was done with tiny numbers of animals being compared and thus had extremely low statistical power. **2).** Sambucci et al. (2010) also had a tiny numbers, with 11 or 12 per group studied in Table 2, from 6 to 35 studied in Table 3 and 6 to 12 studied in Table 4. The claims of no statistically significant effects in Figs. 2, 3, 4 and 5 are based on the tiny numbers in Table 3, are therefore, based on studies with very low statistical power. **3).** The first part of the Aït-Aïssa et al. (2010) paper focused on GFAP values, a measure of gliosis, which is a risk factor for glioma formation. The groups studied in Fig. 4 of Aït-Aïssa et al. (2010) range from 3 to 10, so again we have tiny numbers and the authors report that none of the exposures, SAR = .08, = .4. or = 4 W/Kg produced statistically significant changes according to their statistical calculations. As in the other studies, no raw data are provided but Fig. 4 provides bar graph information which includes median values for each of the 10 different regions of the brain in these rats, control rats and also rats exposed either pre-natally or both pre-natally and post-natally. For 5 of those brain regions, M4, CA1, CA2, CA3 and DG, the median values are high enough that one can see which are higher and which are lower from the graph. It appears to this author that the median values go up from the sham exposures to the lowest intensity (= .08), that they drop going to the next intensity (= .4) and that they go up going to the highest intensity studies (= 4). You may recall (see above) that there are certain windows of exposure that give the highest biological response but with both lower and higher intensities giving lower responses. It follows that the complex apparent dose-response curve of Aït-Aïssa et al. (2010), can be explained by these window effects. The question is whether any such apparent changes are statistically significant? I did, therefore a Chi-square analysis of these data, to determine statistical significance, using both the only prenatal and both prenatal and postnatal exposures (see Fig. 4 in Aït-Aïssa et al., 2010). Those data show that in 10 out of 10 cases, the median value increased going from sham to .08 comparing the sham values with the values at 4 ($P < .02$). It follows from this, that three of the comparisons show statistically significant changes, and the fourth falls just short of statistical significance. Does this mean that that we should conclude that Wi-Fi can cause gliosis and thus possibly gliomas? No, but only because they did not study Wi-Fi. It should be noted, however that the long-term effects on the brain from pre-natal exposures may be relevant to autism causation.

4). Poulletier de Gannes et al. (2012) also suffered from tiny numbers in their study, with 12 to 15 rats studied in each group in Fig. 1, only 5 females in each group in Table 1, 12 to 15 rats in each group in both Table 2 and Table 3. **5).** Aït-Aïssa et al. (2012) also suffers from tiny numbers of rats in the various studies. It used from 9 to 12 pregnant female rats in each group to attempt to assess EMFs impact of reproduction; it used 9 to 12 juvenile rats to determine if EMFs act to change antibody production; it used 9 to 12 young rats to determine whether EMFs impact growth over time. These tiny numbers mean that failure to find statistical significant changes has very low power to support any inferences. **6).** Aït-Aïssa et al. (2013) had similar problems with tiny numbers, 6 to 12 in Fig. 5, 5 to 11 in Fig. 8 and 6 to 12 in Fig. 9. **7).** Poulletier de Gannes et al. (2013) also suffers from tiny numbers. Fig. 1 groups each had 12 males or females and there were also groups of 12 studied in Table 1, Fig. 2 and Table 2. Regarding, the authors give no information regarding statistical significance or lack thereof; rather they only state that the values of these groups were "similar", without providing a definition of "similar". However in comparing the values of testis weight and epididymis weight at 4 W/Kg exposure vs sham control, they provided values for the mean and standard error of the mean (SEM). It is usually the case that when the mean values differ by more than 2.4 times the SEM, the difference is statistically significant. Here the testis weight, comparing sham with 4 W/Kg, values differed by 3.18 times the SEM and the epididymis weight differed by 3.40 times the SEM, each arguing strongly for statistical significance. This raises the question of why the authors failed to provide their P values?

An additional flaw of these 7 supposed Wi-Fi studies is that they each studied exposures of 2 h per day, 5 days per week except for one that only studied one hour per week, 5 days per day. Given that many people are exposed to Wi-Fi fields for 5, 6, 8 or more hours per day, this is another factor which argues that these studies may have been set up to minimize any effects seen.

To sum up the other flaws:

1. The 6 antennae of the reverberation chamber used in 6 out of 7 studies, minimized probable effects produced through the arrangement of the antennae in such a way as to greatly lower the polarization of the EMFs.
2. The use of 1 mm aluminum wires to produce the reverberation reflections, further decreases such polarization, again lowering probable effects. These structures are clearly very different from those found in genuine Wi-Fi, emphasizing the point that these are not genuine Wi-Fi studies, because of 1 and 2 here.
3. Differences in path lengths for different photons, produced by reverberation produce substantial destructive interference.
4. Furthermore the EMFs fed into the antennae are not genuine Wi-Fi either. It follows from this that claims that these are studies of genuine Wi-Fi made by both the authors of these individual studies and by Foster and Moulder (2013) are false.
5. The claims made by Foster and Moulder (2013) that there are no effects produced are also false; the most that may be legitimately concluded is that there is no statistically significant evidence of effects.
6. Each of the 7 studies used only tiny numbers of animals in each

($P < .002$.). Similarly, in 10 out of 10 cases, the median value drops going from .08 to .4 ($P < .002$). However in 8 out of 10 cases, the median value increases going from .4 to 4 ($P < .07$), falling just short of statistical significance. The median values increased with exposure, group studied, such that lack of statistical significance, because of the low power of these studies, drastically limits the drawing of inferences.

7. Finally, 3 out of 7 had evidence of statistically significant effects,

with each of these being ignored by Foster and Moulder.

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